UNITED STATES BANKRUPTCY COURT
DISTRICT OF DELAWARE

IN RE:	.	Case No. 01-1139 (JKF)
	.	
W.R. GRACE & CO.,	.	
et al.,	.	USX Tower - 54th Floor
	.	600 Grant Street
	.	Pittsburgh, PA 15219
Debtors.	.	
	.	March 25, 2008
. . . . .	.	9:09 a.m.

TRANSCRIPT OF TRIAL
BEFORE HONORABLE JUDITH K. FITZGERALD
UNITED STATES BANKRUPTCY COURT JUDGE

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I N D E X

THE COURT: Good morning. This is a continuation of the evidentiary hearing on the personal injury estimation trial in W.R. Grace 01-1139. Participants by phone, Andrew Chan, Beau Harbour, James Wehner, Francis Monaco, Andrew Hain, David Turetsky -- Turetsky, I'm sorry, Jonathan Alden, Alan Madian, John Green, David Parsons, Debra Felder, Andrew Craig, William Wagner, Matthew Kramer, Daniel Speights, James Rieger, Alex Mueller, Lewis Kruger, David Beane, Ari Berman, Shayne Spencer, Guy Baron, Walter Slocombe, Bernard Bailor, Elihu Inselbuch, Jeanna Rickards, Peter Lockwood, Mark Hurford, Leslie Kelleher, Michael Lastowski, Christima Kang, Robert Horkovich, Catherine Chen, Janet Baer, John Phillips, Marti Murray, Jason Solganick, Brian Mukherjee, John Ku, Tiffany Cobb, Nathan Soucy, Theodore Tacconelli, Matthew Daiker, William Corcoran, Michael Scott, Matt Doheny, Michael Davis, Jonathan Brownstein, Darrell Scott, Elizabeth Devine, Scott Baena, Timothy Cairns, Martin Dies, Jay Sakolo, Edward Westbrook, Natalie Ramsey, Kim Christensen, Ken Pasquale, Peter Shawn, Katharine Mayer, William Sparks, Terence Edwards and Christina Skubic.

Have any parties changed in court this morning entries from yesterday? All right, we'll just proceed --

MS. HARDING: Well, actually, Your Honor --

MR. STANSBURY: Brian Stansbury for W. R. Grace.

THE COURT: Anyone else changed? I'm sorry. I can't see anybody behind you.

MS. HARDING: Scott McMillan is not here at counsel table for Grace, Your Honor.

THE COURT: Anyone else? Okay. Thank you. Ms. Harding? Oh, actually we've got a court reporter change and she may not know you, so maybe I better have you enter appearances. Cathy, we do need to do that. I'm sorry. I apologize. We do need to enter appearances. One second please, let me get -- thank you, go ahead.

MS. HARDING: Barbara Harding on behalf of Grace.

MR. BERNICK: David Bernick on behalf of Grace.

MR. STANSBURY: Brian Stansbury on behalf of Grace.

MR. FINCH: Nathan Finch on behalf of the ACC.

MR. BAILOR: Bernard Bailor on behalf of the ACC.

MR. INSELBUCH: Elliot Inselbuch on behalf of the ACC.

MR. MULLADY: Ray Mullady for the FCR.

MR. ANSBRO: Good morning, Your Honor, John Ansbro for the FCR.

MR. HOROWITZ: Good morning, Your Honor, Greg Horowitz on behalf of the equity committee. I will be on the posters today.

MS. KRIEGER: Good morning, Your Honor. Arlene Krieger from the Official Committee of Unsecured Creditors.

MR. KRAMER: Good morning, Your Honor. Matt Kramer on behalf of the Property Damage Committee.

MR. FRANKEL: Good morning. Roger Frankel on behalf of the FCR.

THE COURT: Is that it? Okay, Ms. Harding.

MS. HARDING: Good morning, Your Honor. Grace would call Dr. Suresh Moolgavkar to the stand please.

THE COURT: All right. Will you please spell that for the record please, Dr. Moolgavkar's name?

MS. HARDING: Yes, Suresh, S-u-r-e-s-h, Moolgavkar, M-o-o-l-g-a-v-k-a-r.

THE COURT: Thank you.

S U R E S H M O O L G A V K A R, WITNESS, SWORN

DIRECT EXAMINATION

BY MS. HARDING:

Q Good morning, Mr. Moolgavkar.

A Good morning.

Q Pull the mic down a little bit more. Could you state your name for the record please?

A Yes, my name is Suresh Moolgavkar.

Q Dr. Moolgavkar, what is your profession?

A I am an epidemiologist.

Q How long have you been an epidemiologist?

A For something over 30 years.

Q Did you prepare any slides to assist you with your testimony today?

A Yes, I did.

Q Did you prepare a slide on your professional credentials?
A Yes, I did.
MS. HARDING: Could we see G-2230 please?
Q Where have you spent the majority of your professional life?
A The majority of my professional life has been spent in Seattle at the Fred Hutchinson Cancer Research Center and the University of Washington.
Q What were your academic -- what is your academic appointment at the University of Washington?
A I'm a professor in the Department of Epidemiology and adjunct professor in the Department of Biostatistics and an adjunct professor in the Department of Applied Mathematics.
Q And what is your position at the Fred Hutchinson Research Center?
A I'm a member of the Fred Hutchinson Cancer Research Center which is a title that is equivalent to that of full professor at an academic institution.
Q And what is your current status at both University of Washington and the Fred Hutchinson Cancer Research Institute?
A Currently I am on leave of absence from both the Fred Hutchinson Cancer Research Center and the University of Washington while I have -- from the 1st of April 2007.
Q Okay, so for approximately the past year?
A Yes.

Q I know that -- I understand that in biostatistics and quantitative epidemiology there is a model called the Moolgavkar Benson Knudson model, the MBK model, is that correct?
A That's correct.
Q Is the Moolgavkar that's referred to in that model, is that you?
A Yes, it is.
Q What is your -- if we could go to 2231 please. What is your -- you said you are on a leave of absence. What is your current professional status?
A Currently I work for Exponent which is a large international consulting company. I am the Director of the Center for Epidemiology, Biostatistics and Computational Biology, and I'm also a corporate vice president.
Q Prior to joining Exponent last year had you worked as a consultant with Exponent in the past?
A Yes. Prior to joining Exponent full time on April 1, 2007 I was a consultant. I used to do consulting on the side and some of that consulting I did through Exponent.
Q What kind of consulting work does Exponent do at the center that you direct? And -- I'm sorry, go ahead.
A Yes, I direct the Center for Epidemiology, Biostatistics and Computational Biology. And as the name suggests we do work in epidemiology, biostatistics and computational biology. So

the work involves work both for industry and some work for government. It involves the conduct of epidemiological studies and biostatistical analysis. It also involves some consulting work of the type I'm doing here today; litigation support and so on. But, it involves working with environmental issues such as air pollution and things, and work of that nature.

Q 2232 please. In addition to your work at The University of Washington, The Fred Hutchinson Cancer Research Center, have you had other academic appointments, as well?

A Yes, I've been on the faculties of The John Hopkins University, Indiana University, The University of Pennsylvania and The Fox Chase Cancer Center. By way of explanation The University of Pennsylvania and The Fox Chase Cancer Center have very close collaborations and affiliation agreements. Both are located in Philadelphia. And then --

Q Is that the same type of arrangement that The Fred Hutchinson Center has with The University of Washington?

A Pretty similar, yes. And so for the last 24 years I have been with The Fred Hutchinson Cancer Research Center and The University of Washington.

Q Okay. How many years did you spend at The Fox Chase Cancer Center and The University of Pennsylvania?

A I was there for seven years.

Q I think you've already talked about your title at University of Washington.

Q 2233 please. Dr. Moolgavkar, have you been invited to appear on various panels around the world with respect to the issue of biostatistics and epidemiology?

A Yes. I've been on various panels and working groups. I have been on several panels for IARC which is the International Agency for Research on Cancer. That is the cancer research arm of the World Health Organization. I was a member of the working group on the monograph for tobacco smoking. I was a member of the working group and senior editor of the monograph on quantitative estimation and prediction of human cancer risk.

Q Slide 2233. Does it list many of the panels that you've appeared on?

A Yes, it does.

Q Okay. You didn't want to put it in a slide, but I understand that you've also received several awards for your work in the field of quantitative epidemiology, is that right?

A That's correct.

Q Could you tell us what the founders award was?

A The founders award was given to me by the Chemical Industry Institute of Toxicology Centers for Health Research in North Carolina for my work on developing the MBK model.

Q And previously I understand there were other eminent scientists that have received that award?

A Yes. That award was also given to Dr. Henry Peto who was the director of the McCardel Lab at the University of

Wisconsin. It was given to my colleague Alfred Kinutsin. It has also been given to Sir Richard Gall an eminent epidemiologist.

Q Have you also been an editor for several important peer review journals?

A Yes. I have served on the editorial board to several journals.

Q Okay. Sorry, 2234. Does this list some of the journals on which you have been an editor?

A Yes. I am currently an associate editor of "Risk Analysis." I'm on the editorial board of "Inhalation Toxicology" and on the editorial board of "Biology Direct." I served for about half a dozen years as an associate editor of "Genetic Epidemiology." And I am the senior editor for three volumes, three books that is on epidemiology biostatistics and risk assessment.

Q Actually that was my next question. I wanted to ask you about the books. So you have been an editor on several books as well?

A Yes.

Q What is the role of an editor of a volume or a book of the type that you work, you described there?

A These books actually were articles that arose from a meeting, a week long meeting that I had organized on the topics of epidemiology, biostatistics and risk assessment. And the

role of the editor there is to invite the speakers and to solicit chapter contributions from them, to see that they are properly being reviewed and then to either accept or reject those articles for publication in the book.

Q With respect to the books that you've edited, you said you organized the panels, who asked you to do that?

A Well the organizers of the conference asked me to do that and most of these conferences were, I believe they were funded at least in part by the Department of Energy. And so this was more than 10 years ago so I don't recall all the details, but the funding came from the Department of Energy I believe.

Q Could you go to 2236 please? Does 2236 list the journals in which your publications have appeared?

A Yes, that is correct.

Q And about how many published -- how many papers have you published in the field of biostatistics and quantitative epidemiology?

A I've published approximately 150 papers in those fields. About 26 of them directly involve cohort or case control studies and 18 involve analysis of registered data.

Q Now, Dr. Moolgavkar, I know I have heard you previously say I am not an expert on asbestos, what do you mean by that?

A What I mean by that is that I am not an expert on the mineralogy or the chemical properties of asbestos. I'm not an expert on the electron microscopy of asbestos. I'm not an

industrial hygienist. I don't know how to measure asbestos fibers. But, I do consider myself an epidemiology (sic) in the area of asbestos epidemiology.

Q I think you meant expert.

A I do consider myself an expert in the area of asbestos epidemiology, yes.

Q Have you listed on Slide 2237 your experience in the area of fiber carcinogenosis?

A Yes, I have.

Q Could you just briefly talk a little bit about -- you've got several papers on fiber carcinogenosis, what do those generally relate to?

A Well, one of the topics of interest to me over the years has been how biopersistence -- that means how long fibers persist in the body -- how that is related to the toxicity of the fiber. So these are five papers that I wrote on the subject of biopersistence. Basically the central theme of these papers is the role of biopersistence and fiber length in determining the toxicity of the fibers.

Q Okay. You were also an invited expert by WHO at a workshop. What is WHO?

A That's the World Health Organization.

Q And what was the discussion topic at that workshop?

A Well, it was a workshop on mechanisms of fiber carcinogenosis. So some of the basic biological properties of

fibers and how they are toxic. The mechanism by which they are toxic were discussed, as well as an assessment of substitutes for chrysotile asbestos to see whether there are -- whether the manmade fibers are less or more important or less or more toxic than chrysotile.

Q And were you also invited by the Chrysotile Institute to give a -- to participate in a panel there, as well?

A That is correct.

Q Okay, and was that related to the same issue you just discussed?

A Yes, it was. I basically discussed the epidemiology of asbestos and cancer and also some of my own work on fiber biopersistence.

Q It says here that you have an extensive review of the asbestos literature. About -- if you can quantify it -- about how many publications in the field of epidemiology have you read relating to asbestos and carcinogensis?

A I would say scores or hundreds of papers in asbestos epidemiology.

MS. HARDING: Could you put up 2238, please?

Q Dr. Moolgavkar, how would you characterize your primary scientific research and investigative work at The Fred Hutchinson Cancer Research Center?

A I would say that if there is one central theme in my research is the understanding of the relationship between

fundamental biological processes occurring in the cell and the epidemiology of human cancer. That I would say is my central interest. So I'm interested in seeing how events occurring at the level of the cell such as mutations and pertubations of cell proliferation affect cancer rates in human populations.

Q And have you also developed models and methods for understanding cancer risk?

A Yes. As a part of that process of understanding the link between cell biology and cancer I have developed mathematical models that I've used for the analysis of epidemiological data in human populations. So that involves the development of the appropriate statistical tools and the computational tools for doing such analysis.

Q And what is the IARC monograph that is up on the slide here?

A Well this is a monograph in which I was a senior editor and dealt with the topic of quantitative estimation in prediction of human cancer risk.

MS. HARDING: Your Honor, at this time I move that Dr. Moolgavkar be accepted as an expert in the field of biostatistics and quantitative epidemiology.

MR. FINCH: Voir Dire, Your Honor?

THE COURT: Yes.

VOIR DIRE EXAMINATION

BY MR. FINCH:

Q Good morning, Mr. Moolgavkar.
A Good morning.
Q My name is Nathan Finch. I represent the Asbestos Claimants Committee in this case. You are not licensed to practice medicine, are you, sir?
A No, I'm not.
Q You are not board certified in any medical speciality, correct?
A That's correct.
Q You have a PhD in mathematics, correct?
A That's correct.
Q You don't have a professional degree in epidemiology, correct?
A No. I have post doctorate training, but no professional degree.
Q You have never conducted any studies in asbestos epidemiology, have you?
A No, I'm not.
THE COURT: I'm sorry. He has not done what? I apologize.
MR. FINCH: He has never conducted any study in asbestos epidemiology.
Q You haven't done that.
A That's correct.
Q You've never designed an epidemiological study looking at

asbestos as a cause of disease, correct?
A That's correct.
Q None of your publications relate to asbestos as a cause of mesothelioma, correct?
A No, not mesothelioma.
Q None of your publications relate to asbestos fibers, isn't that also correct?
A Well, there is at least one publication that compares the biopersistence of asbestos fibers and its toxicity with the other manmade fibers.
Q But, it doesn't compare that specifically with reference to mesothelioma, correct?
A No, with reference to lung cancer.
Q You have never diagnosed anyone with mesothelioma?
A That's correct.
Q And the first time you started to do any work with respect to asbestos as a cause of disease was around 2000 or 2001?
A Well I knew the general literature before then, but I got involved more deeply in it around 2000 or so.
Q And you first got involved more deeply in it when you were hired by W.R. Grace Company in an EPA cleanup action, correct?
A That's right, yes.
Q And over the past five or six years you worked for something called Exponent and the vast majority of your work related to asbestos as a consultant for automobile companies,

correct?
A That is not correct. I have not worked for Exponent for the past five or six years.
Q Okay, you worked for Exponent for the past few years -- three years, correct?
A No that's not correct either. I worked for Exponent. I've been -- I was a consultant to Exponent for a few months before I joined it full-time which was on April 1 of 2007.
Q Is it correct that since 2001 you have worked primarily as a consultant for car companies and W.R. Grace in asbestos matters?
A Yes, that would be correct.

MR. FINCH: Your Honor, we have a Daubert motion with respect to Dr. Moolgavkar to the extent that he is offering any opinion, a threshold opinion, that some level of exposure to asbestos cannot cause mesothelioma. We believe that is outside of mainstream science and we have filed Daubert papers with respect to that. I don't think he's qualified to offer an opinion that any level of asbestos exposure cannot cause mesothelioma.

CONTINUED DIRECT EXAMINATION

BY MS. HARDING:
Q Dr. Moolgavkar, how long have you been a professor of epidemiology?
A For more than 30 years.

MS. HARDING: And I'd like to -- may I approach, Your Honor?

THE COURT: Yes.

Q I'd just like to point you to this first exhibit that we haven't actually talked about yet. It's called the scientific method. Do you see that?

A Yes, I do.

MR. FINCH: Your Honor, you haven't ruled on the Daubert motion.

THE COURT: She's cross examining with respect to voir dire.

Q And, Dr. Moolgavkar, what are the methods that epidemiologists use to understand risk of disease regardless of the disease and regardless of the potential carcinogen?

A Well, I think the two most important elements in coming to any epidemiological conclusions is first the development of a hypothesis and then the design and the conduct of property controlled epidemiological studies. That is absolutely fundamental. It's the central theme in drawing any conclusions from epidemiological research, and it cuts across all kinds of exposures whether it is asbestos, or radiation or arsenic. The same basic epidemiologic methods are used to study and investigate the issue of the association between the environmental agent and the disease in question.

Q And have you spent the last 30 years of your life

investigating potential carcinogens in the environment?

A Not only have I spent the last 30 years of my life investigating carcinogens in the environment, a lot of my focus has been on developing models and methods for low dose risk extrapolation. So that I'm very familiar with all the methods that are used to investigate risks at low exposure to environmental agents.

MS. HARDING: Your Honor, I again move for Dr. Moolgavkar to be accepted as an expert in the field of biostatistics and quantitative epidemiology.

THE COURT: Well I think he is qualified to express an opinion in those areas, whether or not that area encompasses expressing an opinion with respect to whether or not a person can develop mesothelioma based upon a particular level of exposure, I don't know yet. So I think within the broad parameter of his expertise he's certainly qualified in the field of biostatistics and epidemiology. There is, I think, no question with respect to that. Whether we get to the ultimate conclusion I think remains to be seen.

So at this point I don't know. I haven't heard enough to know. Raise the objection again when the opinion is offered and by then I will have heard the testimony and I will be prepared to rule. But with respect to the gentleman's qualifications, he's clearly qualified to offer opinions within the field of his expertise.

MR. FINCH: Thank you, Your Honor.

BY MS. HARDING:

Q Dr. Moolgavkar --

MS. HARDING: I'm sorry, could you put up 2239 please?

Q Could you describe what you were -- you summarize what you were asked in this case?

A Yes. First I was asked to review the epidemiological data on asbestos and human disease and to identify the diseases that have been demonstrated by scientifically rigorous methods to be associated with or caused by asbestos exposure.

I was also asked to look at dose response relationships for asbestos associated diseases and to draw conclusions regarding the effects of asbestos exposures at various doses in humans, and finally to note what these dose response models might have to say for asbestos related doses below the range of observations in epidemiological studies.

Q I have actually listed those three things here on this sheet here so we can just kind of keep track.

MR. FINCH: Your Honor, can I see that?

THE COURT: Yes, certainly.

MS. HARDING: It's the same thing as the study -- as the slide.

MR. FINCH: Oh. Okay.

MS. HARDING: I just don't want to have to keep

coming back to the slide.

MR. FINCH: Oh.

MS. HARDING: I just wanted to write it here so we know what we're going to talk about.

MR. FINCH: Oh, all right.

Q With respect to those three questions, Dr. Moolgavkar, or those inquiries, and let's focus first on the first one, the review of epidemiological data identified diseases demonstrated scientifically to be caused by asbestos exposure. How does the field of epidemiology, or how do epidemiologists, scientists, answer that question and the other questions that you been asked to address today?

A Yes. As I said earlier the first step in the scientific method is to formulate a hypothesis and then to design properly controlled studies to investigate that hypothesis. In epidemiology generally one positive study only suggests that the disease might be associated with the exposure. The study needs to be repeated several times and if consistent results are obtained in all the studies then you might conclude that the disease is causally associated with the exposure.

One corollary of that is that the totality of the epidemiological evidence must be taken into account when drawing any conclusions regarding the association of an exposure with the disease.

Q Dr. Moolgavkar, will your testimony here today be limited

to opinions based upon a reasonable degree of scientific certainty based upon scientific methods that you have employed throughout your life in all of your areas of investigation?

A Yes. Insofar as any conclusions regarding the association of asbestos to disease in the range of observations is concerned that opinion will be offered with reasonable degree of scientific certainty. But there are areas of exposure called putative exposure or presumed exposure with no observations at all in the epidemiological literature. And in that case mathematical modeling or extrapolation procedures have been used, and here the findings are much less certain.

Q Now, when you say no epidemiological observations at all, by that do you mean no particular study or no reliable body of epidemiological data to allow you to reach a conclusion?

A Well, there is no reliable body of data on exposures that might allow you to come to conclusions regarding an exposure response relationship at very low exposure levels.

Q Now, with respect to the very first question here, the review of the epidemiological data and the identification of diseases demonstrated to be caused by asbestos, what have numerous case control and cohort studies investigated that question concerning asbestos exposure in cancers?

A Yes, they have.

Q And what have those studies found?

MS. HARDING: Could you put up 2240 please?

A Yes. I think there is little doubt epidemiological studies have shown that asbestos increases the risk of mesothelioma and lung cancer at sufficiently high doses. I don't think there is any question about that.

Q What about other cancers?

A Well, there is some concern about other cancers, as well. And there is an indication there was a recent study by The Institute of Medicine that concluded that the range of cancer was probably caused by asbestos exposure.

MS. HARDING: Okay. Could you put up 2241 please?

A Yes. This is a summary of The Institute of Medicine study.

Q Does this Slide 2241 reflect the results of The Institute of Medicine's review of the epidemiological data concerning asbestos exposures and other cancers?

A Yes, it does.

Q Okay. What did they find with laryngeal cancer?

A Well with respect laryngeal cancer they found that the evidence was sufficient to conclude that asbestos was causally associated with laryngeal cancer.

Q Okay. And do you generally accept that opinion?

A Well, there are other strong risk factors for laryngeal cancer, namely smoking and alcohol, and in my opinion the evidence still falls short of being sufficient, but I would be willing to accept that laryngeal cancer is caused by asbestos

exposure.
Q What type of epidemiological data were reviewed by the IOM when they investigated these other cancers? They have laryngeal cancer, pharyngeal cancer, stomach cancer, colorectal cancer and esophageal cancer, correct?
A Yes.
Q What was the data that the -- what was the epidemiological data that The Institute of Medicine reviewed?
A Yes, they reviewed the epidemiological data both what are called cohort and case control studies to come to these conclusions.
Q With respect to pharyngeal, stomach, colorectal and esophageal cancers, the IOM concluded that the evidence was suggestive of inadequate, is that correct?
A Well for esophageal cancer it was inadequate, but for the other three that you mentioned pharyngeal, stomach, colorectal there was suggestive evidence that The Institute of Medicine Committee decided that there wasn't sufficient evidence to conclude a causal relationship.
Q Okay. And in connection with those cancers, what types of studies were reviewed by the IOM?
A The same types of studies that they reviewed for laryngeal cancer; cohort and the case control studies.
Q And indeed with respect to each of those other cancers where the IOM found that there was insufficient evidence and

that the evidence was inadequate, does that mean that there were no studies that they reviewed where the investigators found a statistically significant positive association between those cancers and asbestos exposure?

A Not necessarily. What the committee made the decision on, they based that decision on the totality of epidemiological evidence of each one of these cancer sites.

Q Indeed, if you were to go out into the literature or into the world into hospitals or anywhere, you would find people, individuals, who are -- who have pharyngeal, stomach, colorectal cancer and esophageal cancer who have been exposed to asbestos, correct?

A That's correct.

Q And indeed you might even have case reports that people that have those cancers have been exposed to large amounts of asbestos indeed or low amounts, correct?

MR. FINCH: Object to the leading, Your Honor.

THE COURT: It is leading.

Q Dr. Moolgavkar, would there -- would you find in the population or in the literature potentially case reports or cases of disease in those areas where individuals have been exposed to asbestos?

A Yes.

MR. FINCH: Objection, leading, Your Honor.

THE COURT: It's still leading.

Q Are there case reports involving asbestos exposure and pharyngeal, stomach, colorectal, esophageal cancer?
A Yes, there are.
Q Okay. And, doctor, on the steps in the scientific method where do case reports falls in the scientific method, in scientists' understanding of this issue; carcinogensis?
A I would say in the very first step. The case reports are observations that might lead to the development of a hypothesis.
Q Are they ever, ever considered design -- a controlled study?
A No.
Q Okay. Dr. Moolgavkar, simply because the IOM found that there was sufficient evidence that laryngeal cancer can be caused by asbestos, does that mean that it can be caused by any level of asbestos exposure?
MR. FINCH: Objection, leading.
THE COURT: It's leading.
Q Dr. Moolgavkar, did the IOM, Institute of Medicine, when it found that laryngeal cancer, that there was sufficient evidence for the presence or absence of a causal relationship to asbestos, did they address the issue of the levels of exposure that could cause that disease?
MR. FINCH: Objection, leading.
THE COURT: Ms. Harding, it is leading. You are

asking him every question that can be answered yes or no rather than letting him answer the question. You are assuming an answer and asking him a question that can be answered yes or no. So why don't we get to the point where he's answering the questions?

Q What did the IOM report with respect to the levels of asbestos exposure that are sufficient to cause laryngeal cancer?

A Well the IOM did not have any specific levels of exposure to asbestos that would cause laryngeal cancer, but these cancers are found in cohorts with very high levels of exposure.

Q Dr. Moolgavkar, I'd like to move now to the second question that you were asked to address which relates to the quantifying the levels of asbestos exposure observed at epidemiologically caused disease?

A Yes.

Q In this litigation do you have an understanding of the relevance of the quantification of asbestos exposures that have been demonstrated reliably to cause risk?

A Yes.

MR. FINCH: Lack of foundation and leading.

THE COURT: This is a foundation question so it's clearly appropriate, but I missed part of it. Would you restate it for me please?

MS. HARDING: Actually I'm going to ask an earlier

question first.
Q From a public health or regulatory perspective, why is it important to understand the -- or, is it important to understand the levels of asbestos exposure that have been demonstrated to cause risk, and if so, why?
A Yes, it's important to understand the levels of any environmental agents that have been demonstrated to cause risk because after all one of the goals of environmental epidemiology is to protect the public against environmental agents. And so, it is important to understand what the risk might be to agents that occur commonly in the environment and to which people might be exposed to. So for to understand that it is not sufficient to understand what the exposure does at high doses. It is also important to try and understand what it does at low doses.
Q In this litigation do you have an understanding of the relevance of the quantification of asbestos exposures that have been demonstrated reliably to cause risk?
MR. FINCH: Objection to the extent she's asking what's relevant and not relevant in the context of litigation.
THE COURT: I can't hear you, Mr. Finch.
MR. FINCH: Objection to the extent she's asking what's relevant and not relevant in the context of this litigation. He can offer opinions about epidemiology but not what's relevant to this case.

THE COURT: She's only asked him whether he has an understanding of the relevance. That objection is overruled.

A Yes. My understanding is that the --

THE COURT: You can only answer yes or no to this question, doctor. I'm sorry.

A Okay.

Q What is your understanding, Dr. Moolgavkar?

A My understanding is that claims have been filed against Grace by claimants who allege exposure to Grace products at various concentrations. And therefore clearly the understanding of what these levels of exposure how they might be associated with the diseases that asbestos is known to cause at high doses is clearly important.

Q Do you have an understanding that Dr. Anderson, Dr. Betty Anderson -- do you know Dr. Betty Anderson?

A Yes, I know Dr. Betty Anderson.

Q Okay. How do you know Dr. Anderson?

A Well she is also a member of the Exponent staff.

Q Do you have an understanding that Dr. Anderson has relied upon some of your estimates and analysis for understanding potential risk of exposure to asbestos and disease?

A Yes.

Q And what is that understanding?

A My understanding is that she has taken some of the calculations that I have done and used them in her own work.

Q And, Dr. Moolgavkar, I'd like to talk about those analysis and those estimates now, okay. And you and I together prepared a board to discuss the various aspects of those analysis, is that reflected in front of you there? I can't read the number from here -- GG-2262?

A Yes.

Q Okay. Let's --

THE COURT: Ms. Harding, is there a binder that has all these exhibits in them somewhere?

MS. HARDING: Yes, Your Honor. I'm sorry. I did not know that you hadn't received it. I apologize. Sorry.

THE COURT: Thank you.

MR. FINCH: Robert, 2262 is not in my binder. Do you have an extra copy of it so I can --

MS. HARDING: I'm sure we do somewhere. I'm sorry.

Q Okay, Dr. Moolgavkar, first I'd like to talk about we're in this area here about quantifying the levels of asbestos exposure observed epidemiologically to risk. And I want to talk first about lung cancer. And are there -- who are the primary researchers who have investigated the dose response relationship between lung cancer and asbestos exposure?

A Well, I would say that since the early to mid-1980s there have been three very important publications that have appeared since that time. One is the "Nicholson Study" which was done for the Environmental Protection Agency and I believe that

appeared in 1986. A second extremely important publication appeared in 2000, that is a paper by Hodgson & Darnton which in some ways is one of the most important papers to have appeared in the study of the dose response relationship between asbestos and mesothelioma and lung cancer.

And the third study that appeared in 2003, it was a study commissioned by the EPA and was authored by Berman and Crump.

Q Have you prepared a slide that describes -- that lists the studies that have been relied upon by the EPA and Hodgson & Darnton to investigate the dose response relationship between lung cancer and asbestos exposure?

A Yes, I have.

MS. HARDING: Okay. Could you show us 2246 please?

Q Are these the studies, Dr. Moolgavkar?

A Yes. In the left-hand column you have all the studies that were looked at by Hodgson & Darnton. These are all cohort studies. There are no case control studies in this group of studies. And the studies marked in red were the ones that Nicholson and the EPA considered in 1986.

Q Okay. And what have these studies found with respect to levels of asbestos exposure?

A Well, first of all, these are cohort studies and Hodgson & Darnton report the average cumulative exposure in these cohorts. The first thing that one notes is that the exposures

are extremely high. The cohort with the lowest exposure, that is the Sweden Cement Manufacturers cohort had a cumulative exposure level of about 13 fiber per mil years. So that's a pretty high exposure.

The other cohorts had extremely high exposures. So that's the first thing one can say. And a corollary of that observation is that any kind of a dose response relationship that can be actually investigated in the range of observations can only be investigated in this high dose cohort. So, there is no direct information on exposure response or dose response relationships below this level of cumulative exposure.

Q Dr. Moolgavkar, going up to the board here to 2262 there is a circle here at 15. How does that relate to the testimony you just provided?

A That circle at 15 is the approximate lowest observed average exposure in the cohorts or in the studies that have been used to characterize the dose response relationship both for lung cancer and mesothelioma.

Q Can you tell us if there is -- is there a reliable body of epidemiological data supporting the conclusion that there is significant risk at levels below that in the literature?

A There is no body of data that supports a significant risk below that level of exposure.

Q When you say there is no body of data, are you saying that there are no epidemiological studies out there that don't

report a statistically significant risk at a lower level?
A No, I'm not saying that. I should be more precise. There might be individual studies that indicate a significant risk, but if you look at the totality of evidence there is no evidence of an increased risk below that level of exposure.
Q You said, I think a little bit earlier, that the studies that are used by these investigators to understand dose response or cohort studies. Why are they cohort studies?
A Well the EPA, Hodgson and Darnton and Berman and Crump all have criteria for using studies for the dose response analysis. These criteria have to do with the -- basically with the availability of reasonable to good exposure information on asbestos exposures. And these were the studies that they selected that met their criteria.

These are cohort studies. One of the characteristics of these studies is these are occupational cohorts so they are workers who are exposed to asbestos in a pretty controlled environment. Their exposures can be more precisely measured than the exposures, for example, in case control studies where workers might move from one job to another. So I think that was one of the reasons that they chose these cohort studies. Exposures were available so that an exposure response relationship could be performed.
Q With respect -- I know this slide is difficult to read, it's small. But, in looking at the cohorts that were studied

and the asbestos in the workplace there, what type of work environments are represented by these cohorts?

A Many of them are mining and milling cohorts in which asbestos was mined or milled. There are textile cohorts, there are cement workers cohorts and so on. Many of these cohorts are cohorts in which there was fairly clear indication of exposure primarily to asbestos and not to other kinds of fibers.

Q Now I'd like to move onto the discussion, still in this section here quantifying the levels of asbestos exposure observed epidemiologically to cause disease and ask you about mesothelioma. Similar to lung cancer, what types of studies are used to investigate the dose response relationship between mesothelioma and asbestos exposure?

A The investigation of the dose response relationship for mesothelioma and asbestos exposure has also been done primarily in cohort studies. There have been some attempts to look at dose response relationships in case control studies. But in my opinion they fall short because of a number of methodological problems.

MS. HARDING: Is this GG-2246? You already have that up there? Okay.

Q Is this the same list of studies that you -- that were used by the researchers in the lung cancer context?

A Yes, these are the same studies that were used by the

researchers in the lung cancer context. But this is -- these are the -- the cohorts in red here are the mesothelioma cohorts that were studied by the EPA. So the EPA looked at more lung cancer cohorts than mesothelioma cohorts.

Q So the only distinction in substance with respect to 2246 as opposed to I think it's 2244 was the highlighting of the studies that the EPA used in their dose response model?

A That's correct.

THE COURT: Wait, I'm sorry. I'm confused. 2246 -- I thought the reds were the studies that the EPA used with respect to lung cancer, but that's not correct, that's the studies used with respect to meso?

MS. HARDING: Yes, if you go back to 2244, Your Honor. There we go. The title you will see has lung cancer.

THE COURT: All right. We've only seen 2246, 2244 wasn't up before.

MS. HARDING: I'm sorry. I put up the wrong slide then.

THE COURT: All right. Wait til I go back and get my notes corrected.

Q Dr. Moolgavkar, we were talking about lung cancer studies and I thought it was 2244 because there was so much red, but 2244 is the slide that describes the cohort studies that have been used by the EPA and Hodgson & Darnton to investigate lung cancer, is that right?

A That's correct.
Q And the red, what does the red describe?
A In this slide, the red describes the subset of these studies that were used by the EPA in 1986.
THE COURT: All right, so 2244 is lung cancer, but the red still indicates what the EPA used. 2246 is meso and the red indicates what the EPA was looking at.
THE WITNESS: Yes, Your Honor.
THE COURT: Okay, thank you.
MS. HARDING: Just to be clear, Your Honor, 2244 are the studies used by Hodgson & Darnton.
THE COURT: Correct.
MS. HARDING: And the red --
THE COURT: Is what the EPA also looked at.
MS. HARDING: Is what the EPA also used, yes.
THE COURT: It's the same set of studies only 2244 is lung cancer, 2246 is meso. It's just that before it was only 2246 that was up and it should have been 2244.
MS. HARDING: That's right, Your Honor.
THE COURT: Okay. I got it. Thank you.
Q Okay. So going back to 2246 please, to mesothelioma. And the red highlighted slides on 2246, Dr. Moolgavkar, what do they represent just so that the record is clear?
A Yes, those represent the studies that were used in 1986 by EPA to come up with risk estimates for mesothelioma.

Q Okay. And what conclusions can be drawn from this body of epidemiological, if any -- this body of epidemiological evidence and a dose response relationship of mesothelioma and asbestos exposure?

A Well, what this data clearly shows is that high levels of exposure to asbestos are associated with the occurrence of mesothelioma. It also shows quite clearly that the different kinds of asbestos fibers have different potencies so that the amphiboles are a lot more potent than chrysotile in causing mesothelioma. And there is some evidence that that's true also for lung cancer.

Q Is there with respect to mesothelioma, a reliable body of epidemiological evidence that supports the conclusion that there is significant risk of mesothelioma of asbestos exposures below that level of 15 that you've indicated on the chart?

A There is not.

Q Now some of the experts on -- that have appeared for the written reports for the ACC have, you know, oh my goodness, Dr. Moolgavkar, how could you say that? How could you say that that's the case? There are -- there is evidence of exposure lower than that demonstrating a significant risk of mesothelioma. What's your response?

MR. FINCH: Objection, leading.

THE COURT: No, that was not a leading question. She was stating the question saying that the ACC was saying that

there is evidence of exposure below that level and what is his response. That is not leading. Go ahead, doctor, you may answer.

A I believe that the studies on which those conclusions are based are fatally flawed and do not support those conclusions.

Q Now I want to ask you another question, a similar question, a related question. I think I'll go up here.

MS. HARDING: Can you see, Your Honor?

THE COURT: Yes.

MS. HARDING: Can you all see?

Q I want to ask you about something that's what I think is referred to as incremental dose. And if you were talking about asbestos exposures three fiber years and six fiber years or asbestos exposures at 15 fiber years or 18 fiber years and asbestos exposures at 100 fiber years or 103 fiber years.

Is there a body of reliable epidemiological evidence that demonstrates that there is a statistically different risk between exposures, asbestos exposures, at the level of three as opposed to the level of six?

A No. There is absolutely no evidence, no direct evidence to suggest. No epidemiological studies that show a significantly increased risk at those small differences in exposure levels that you have indicated in this black board.

Q Regardless of where the exposure level is?

A Regardless of where the exposure level is. It is entirely

possible theoretically that small increments in dose lead to increments in risk. But, this increment in risk would be so small that it would be undetectable in any epidemiological study.

Q Why is that?

A Because of the -- I think basically for two reasons. One is the difficulties in measuring exposures precisely so because of the difficulties of exposure measurement error, and secondly because of the heterogeneity of human populations. I think any difference, even if there is one, would be completely buried in the noise.

Q In the noise. What do you mean when you say that?

A In the background noise, the statistical fluctuations that you get in disease frequency in any epidemiological study.

Q Now the next slide, 2247, I think I understood that to be a summary of your opinions with respect to low exposures of mesothelioma. Is there any -- does that summarize your opinions or is there anything else that you wanted to say about that?

A No, that pretty much summarizes my -- it pretty much summarizes my conclusions. I say that most inferences at lower exposures are based on mathematical extrapolation which I'm going to discuss later in this testimony. As I've said earlier, epidemiological studies that claim to report risks at low exposure levels have very serious limitations and the

contention that every exposure above background contributes significantly to risk is simply not scientifically defensible.

Q Okay, with respect to --

THE COURT: Wait. Could you stop there for a minute please?

(Pause)

THE COURT: All right, thank you.

BY MS. HARDING:

Q Dr. Moolgavkar, with respect to the last statement you just made, could you explain what you mean by that? Is that what you were just talking about when I asked you the questions about the doses there, or is that something different?

A No, that's part of it. There are some risks that are so trivial that they would never be picked up in any epidemiological study. So I can give examples. If somebody smoked say two packs a day of Marlboro's for 20 years and developed lung cancer but just happened to borrow a Pall Mall from a friend one day and -- a Pall Mall cigarette contributed significantly to his lung cancer risk. It's a similar situation.

Q I'd like to ask you now about the second bullet you have up there, the studies purporting to report risks at low levels of exposure. Did you prepare a slide to discuss that issue?

A I have a slide for that, yes.

Q That's GG-2248, is that right?

A Yes.
Q What -- are these the studies that the others have suggested demonstrate that there is increased risk for mesothelioma at lower levels?
A Yeah. These are the case control studies that have been widely used to suggest that there is a strong dose response relationship for mesothelioma at low levels of exposure. And so these are four case control studies and they all have basically pretty serious limitations. First of all, none of them have any reliable industrial hygiene estimates of exposure and that is actually recognized by the authors of the papers themselves. So, for example, "Iwatsubo", et al., every time they report a fiber per mil a year cumulative exposure, they do it quotes. And at several places in the paper they say they never had any direct measurements, that their measurements were reconstructed by industrial hygienists who also did not have any direct measurements of the products they were looking at.

The second limitation is that these studies, even if they attempt to reconstruct asbestos exposures, have absolutely no information on what type of fiber these individuals might have been exposed to or what mixture of fibers they might have been exposed to. And other than that, they also have some serious methodological limitations insofar as these statistical analyses are concerned. And so I simply don't find that the findings or the conclusions of these studies are credible.

And finally, the last study by Roland Natale (phonetic) which is often quoted is not a full study, at all. It's just an abstract, and I'm unable to evaluate that study because I have not been able to find a full text of that study.

Q With respect to the Category 2 that you have up there, the -- meet the criteria for inclusion of the EPA, Hodgson and Darnton, and Berman and Crump for inclusion in their studies to investigate dose response?

A Yes.

Q Okay. Would these studies, or studies like them, have been able to meet that criteria that are included in those studies?

A Well, they were clearly not included. Obviously, none of these studies were available to Nicholson in 1986 but certainly Iwatsubo was available to Hodgson and Darnton in 2000. And all these, Hans and Natale (phonetic) would have been available to Hodgson and Darnton, as well. All three studies would have been available to Berman and Crump in 2003, but were not included.

Q Why -- aside from the fact that some of them weren't available, are there reasons why they wouldn't have been included?

A Well, I think --

MR. FINCH: Lack of foundation. He didn't author the Hodgson and Darnton or Berman and Crump before the EPA 1986

risk assessment. I don't think he can testify as to what those individuals thought about why they included a study or didn't include a study.

THE COURT: Well, that's true. I think he cannot testify as to why someone else may not have included them, but he can specify what the criteria were.

BY MS. HARDING:

Q Did the EPA, Hodgson and Darnton and Berman and Crump discuss criteria for studies that they included in their analysis?

A Yes. As I said earlier in my testimony, they do have criteria and those criteria have to do with the quality of the exposure assessment. And they don't say specifically why these studies were not included, but it seems to me that these studies don't meet those criteria.

Q Okay. And why don't they meet the criteria?

A As I've said, because they themselves, the authors themselves recognized the limitations of their exposure assessments. They have no direct measurements of exposure.

Q So based on the EPA, Hodgson and Darnton and Berman and Crump, is there a scientific method for determining risk at varying levels of asbestos exposure?

A Yes. There is -- different dose response models have been developed. For lung cancer they use more or less the same models, the EPA, Hodgson and Darnton and Berman and Crump use

very similar models for lung cancer. But, for mesothelioma, Hodgson and Darnton use models that are very different from the EPA and the Berman and Crump model.

Q And would these -- has that method been deployed in studies such as the ones that are on this table to quantify a level of statistical significant risk below that 15 that you have on your chart?

THE COURT: I'm sorry, Ms. Harding, I don't know what method we're talking about. There are several methods. I don't know which one we're talking about.

BY MS. HARDING:

Q Okay. Berman and Crump, Hodgson and Darnton and EPA all investigated the issue of dose response from asbestos exposure and disease, correct, is that right?

A That's correct.

Q And in terms of understanding exposures below the 15 that you've indicated on the chart here, are there reliable epidemiological studies that have been done like the ones that are on the charts and used by Berman and Crump, the EPA, and Hodgson and Darnton to find significant risk below the level that you have on the chart?

A No.

Q Now, Dr. Moolgavkar, I want to ask you about another benchmark you have here on this chart. It says auto mechanic exposure and then over here it says risk not observed and I'd

like to ask you about that. First of all, what -- when you say -- what does the 2.8 represent first, let's start there?

A Well, the 2.8 represents -- is a number taken, is a figure taken from a recent paper, an industrial hygiene paper that suggests or says, that concludes that 95 percent of the auto mechanics were exposed to less than 2.8 fiber per mil years of cumulative chrysotile exposure while working in that profession.

Q And why is the auto mechanic exposure important in your analysis here?

A Well, there has been a great deal of concern that automobile mechanics may be at increased risk of asbestos induced disease like mesothelioma and lung cancer because they deal on a daily basis with friction products like brakes and gaskets and so on that contain small amounts of chrysotile asbestos. So, there has been this concern that NIOSH has had and other agencies have had since the 1980s, at least. And so as a result of this there have been multiple epidemiological studies of mesothelioma to see specifically whether automobile mechanics are at an increased risk of either mesothelioma or lung cancer. And none of those studies has ever shown any evidence of any increased risk. And I think I have a slide for that.

MS. HARDING: Could you show 2249 please?

A Yes, these are just the case control studies of risk of

mesothelioma among auto mechanics. And here I have ten case control studies and, Your Honor, the blue dot represents what is called a relative risk. A relative risk of one means that there is no increase in risk. And the vertical lines through the blue dots represent the so-called 95 percent confidence interval. And if one of the ends of the confidence interval crosses that red line at one, it means that none of these estimates are statistically significant.

So the interpretation of this slide is that there were ten case control studies of the auto mechanics that were conducted and none of these studies found a statistically significant risk different from one. So all the risks are basically one, so no increase or decrease in risk.

Q Dr. Moolgavkar, now you said those are case control studies. How can you use them for dose response information?

A Well, there is no direct dose response information in these studies. They --

Q Then, let me ask you a question before you go on. Are you using the auto mechanic case control studies for the information on dose for auto mechanics?

A Well, I'm not using these case control studies directly, but there is one fairly unique feature of work as an automobile mechanic and that is, namely there is pretty homogeneous exposure. They're exposed to friction products in the garages where they work. This is quite different from say construction

work where a construction worker might move from one area to another and he might be -- he or she might be exposed to different kinds of asbestos fibers at different concentrations in different areas. So this is a pretty unique situation, but here you can study the job, namely auto mechanics, arrive at a conclusion regarding the risk and then use industrial hygiene studies done by others to see what the levels of exposure might have been. And this is precisely what I've presented on this graph here, on this -- the 2.8 represents the result of recently published industrial hygiene study for auto mechanics.

Q Aside from the recently published industrial hygiene study with respect to exposure of auto mechanics, have there been numerous, or are there other industrial hygiene studies relating to auto mechanic exposure in the published literature?

A Yes, there are numerous studies in the published literature and I only have a nodding acquaintance with them because I'm not an industrial hygienist.

Q Okay, and so the data that's used to characterize exposure is not the data from the case control studies, it's data from the industrial hygiene studies, is that right?

A That is correct.

Q I do want to ask you one other benchmark that you have on the slide here -- I'm sorry, on the board -- which is on 2262 which relates to the asbestosis and at 25. Can you tell me what that represents please?

A Yeah, the asbestosis is the most serious non-malignant condition that is associated with asbestos exposure and I think it's widely recognized. I think I have a slide to show this. It's widely recognized that below 25 fiber per mil years asbestosis cannot occur.

Q Could you show 2251?

A So I think there's little disagreement on this point that asbestosis does not occur below an exposure of about 25 fiber per mil years.

Q And what are the sources listed on this slide that support that opinion?

A This is the -- three government bodies. There's the Ontario Royal Commission 1984, Doll and Peto which also undertook this work I think under commission from the UK government, and our own EPA that came to the same conclusion.

Q Now, we've been talking about the dose response relationships of asbestos exposure and mesothelioma and lung cancer that have been observed in the epidemiological data, is that right?

A That's correct.

Q Now I'd like to move onto the last inquiry here and talk about your investigation of the exposure response relationships for asbestos related disease both in the range of observation and in the model derived range of observations. I want to talk about the modeling that's been done with respect to asbestos

exposure and disease. Okay?
A Model derived range of extrapolation you mean?
Q Yes, okay. Are there methods that have been developed to estimate risk at lower levels of asbestos exposure than those actually observed in the epidemiological data?
A Yes. One uses basically mathematical models, mathematical and statistical models to fit the data in the range of observations and then those models are used to extrapolate down to levels where there are no observations.
Q And, Dr. Moolgavkar, is that type of modeling the kind of modeling that you have been working with most of your professional career?
A Yes, that is exactly the kind of modeling that I have been doing for 30 years.
MS. HARDING: Could you put up 2252 please?
Q And just to be clear, we've moved now from risk that's been observed epidemiologically with the body of epidemiological data to support it, is that right?
A That's correct.
Q To the model derived range of extrapolation, is that right?
A Yes.
Q Okay. And are you familiar with this slide?
A Yes. This slide was in one of my reports and it's a schematic that simply shows the process of low dose

extrapolation. So what you have shaded in dark gray on the right-hand side of this chart is the range of doses in the -- that are observed. So in this range you have some range of exposures of doses and you have some observations that is the response or the disease data, the frequency of disease occurrence, for example. And what you can do is fit a mathematical model to those points and then make some decision as to how that risk is to be extrapolated down to the range where there are no observations, at all.

And so there is a range of possible responses and it is generally considered that the linear extrapolation down into the unobserved range is the most protective, is conservative, and the most protective of public health. We have no idea that it is correct, and the further away you get from the observations, the more uncertain the results. But that -- for public health protection, that linearity often assumed by agencies that are charged with protecting the public health, but that doesn't mean that that is the correct model to use. In fact, we don't know what the correct model is in that range.

Q Are there different models that have been developed to fit the data in the observed range to understand risk in the lower range?

A Yes, there are indeed different models that have been developed that fit the data equally well in the observed range but might actually diverge quite widely in the range of

extrapolation.

MS. HARDING: Could you put up 2258 please?

Q Does this slide try to summarize what you believe to be the important factors that the Court needs to understand -- to understand modeling in the unobserved range?

A Yeah, I believe so and I --

Q Could we -- let's walk through each one then.

A Yes.

Q The first one says mathematical models are used to investigate dose response relationships both in the range of observation and the range of extrapolation. I think that just described that process, right?

A Yeah. Well, I like this quote from the statistician G.E.P. Box who said essentially all models are wrong but some models are useful and I think that's the way in which models should be treated. Mathematical models are used to investigate dose response relationships and one has to understand that this is both in the range of observation and in the range of extrapolation. Now, both in the range of observation and in the range of extrapolations, different models yield different answers. So sometimes one is able to choose a model that fits the data better in the range of observations. One would use that model then. But different models can yield different answers in both ranges and what is important to realize is that the further from the range of observations one gets, the more

discrepant the models might become and the less certain the results.

Q I'm sorry, did I interrupt you?

A Yes, I was just going to go through all the points.

Q Well, I was just going to ask you about the next bullet even in the range of observations prediction of monotone increase in risk is based on models, what does that mean?

A Even in the range of observations as I showed on the previous slide, if we could go back to that for just a minute, you can see that in the range of observations you have those circles that represent the responses and some of them lie above and some of them lie below the line that has been fit to it. So the model suggests that there is a monotone increasing dose response relationship; that is, as the dose increases, the response increases, also. That is the model that fits the data -- how it would directly -- the data can fluctuate on both sides of that line.

Could we go back to that slide? Thank you. So this is what I mean. Even in the range of observations, prediction of monotone increase in risk is based on models. And there really is no direct observation that small increases in exposure lead to increase in risk. And I said earlier this just reiterates a point I made earlier that one would not be able to design an epidemiological study that would be able to tease apart a small increase in risk.

Q Let's talk now about the models that have been -- that are available and used in connection with asbestos exposure.

MS. HARDING: Could you show 2254 please?

Q And are these -- does this slide reflect the models that are available in the area of asbestos exposure and lung cancer and mesothelioma?

A Yes, there are two distinctly different types of models used for lung cancer and mesothelioma. For lung cancer, most lung cancers can be analyzed in the cohorts in the observable range of data. And one of the targets of estimation in cohort studies is the relative risk. So the relative risk is directly estimated from the cohort studies. So what the models can do is that they can directly model the relative risk and so the kind of model that is used is called a linear access relative risk model for lung cancer.

Now for mesothelioma, this is not so easy because mesothelioma is a rare cancer and relative risks are not estimated from cohort studies. You cannot estimate relative risks from cohort studies simply because the background risks are so small. So one has to do mesothelioma modeling in a different way. One has to use what are called absolute risk models and there are two that are generally available. There's the Peto model which the EPA used in 1986. This was not a fiber type specific model. Namely, they threw in all kinds of asbestos types, fiber types together in that model in 1986.

And then there's the Berman and Crump model in 2003 which made a distinction between chrysotile asbestos and amphiboles. So these are both versions of what I would call the Peto model.

The second kind of model was developed by --

THE COURT: Sorry, doctor, the Berman and Crump made a distinction between amphibole and what?

THE WITNESS: And chrysotile.

THE COURT: Okay, the two asbestos fibers?

THE WITNESS: Yes.

THE COURT: Thank you. Was it specific to asbestos fibers, that study?

THE WITNESS: Specific to asbestos fibers.

THE COURT: Okay, thank you.

A Hodgson and Darnton developed a model based on average cumulative exposures in cohorts and their model is of a completely different type. It's not the Peto model. It's a completely different type of model.

Q Okay now, with respect to your analysis in this case -- but before we get there, let me ask you this. What does the term "doubling-dose" refer to, have you heard that term and what does it mean?

A The doubling-dose to me indicates the dose at which the relative risk is equal to two. Namely, that's the dose at which you increase -- at which the exposure -- the probability of developing the disease in the exposed population against the

unexposed population is two. That is also the demarcating line if you will between -- about which it is more likely than not that a disease can be attributed to the specific exposure that you're thinking about.

Q Are there methods available in the scientific literature for calculating the doubling-dose observed or extrapolated in the models?

A There's one -- one would use models of this type to calculate the doubling-dose. For lung cancer one would use the relative risk model. For mesothelioma one has to use a two step process which I think I have a slide for that.

Q You do. We're going to get there, I'm asking you about lung cancer first. But have you calculated the doubling-dose estimates for asbestos, both using the models, both in mesothelioma and lung cancer in this case?

A Yes, I have.

Q Okay, let's discuss those estimates.

MS. HARDING: And if you could show 2255 please.

A Okay, so as I said earlier, estimation of doubling-dose, the dose for which relative risk equals to two for lung cancer and mesothelioma depends upon the use of mathematical models. There's other critical difference in that for lung cancer the doubling-dose is directly estimated because the relative risk is directly estimated in cohort studies. For mesothelioma you need a two-step process. The doubling-dose for mesothelioma

then involves applying what is called an absolute risk model and what this model does is calculate or estimate for you the total number of mesotheliomas that you would get for a specific asbestos exposure, but that is the numerator. But, you need a denominator; namely, what is the background risk without exposure to asbestos? So the second step needs some assumptions regarding the background lifetime probability of developing mesothelioma.

Now when you have both these pieces in place, then you can take the numerator, divide by the numerator -- by the denominator and come up with an estimate of the doubling-dose.

Q And is there information that allows you, sometimes at least, to estimate doubling-dose information for different fiber types?

A Yes, the -- both the Berman and Crump version of the Peto model and the Hodgson and Darnton formulas allow you to use -- make distinction by fiber type. I prefer the Berman and Crump formulation. I prefer to use the Peto model.

Q Okay, let's talk first about the relevant -- your relevant doubling-dose estimates for lung cancer, okay?

A Yes.

MS. HARDING: And could you show 2256 please?

Q I understand this to be the results of your estimates for doubling-dose. But, before I ask you about that, did you use the standard methods that are available in quantitative

epidemiology and biostatistics for calculating the doubling-dose of asbestos exposure and lung cancer?

A Yes. Yes, I did. These are fairly standard methods. For lung cancer the so-called linear relative risk model has been used for many, many years and I have laid out the details of how this calculation is done in my reports. But, what I come up with are the following two estimates of doubling-dose. For EPA in 1986 which looked at mixed fibers, the doubling-dose estimate is about 100 fiber per mil years. For Libby miners, based on an estimate of the potency for lung cancer reported in McDonalds' 2004 paper, I estimate the doubling-dose to be about 278 fiber per mil years.

Q With respect to the first number, the EPA 1986 doubling-dose number, what fibers are being -- what kinds of asbestos fibers are being modeled there with respect to doubling-dose?

A I believe that the EPA -- the cohorts chosen by EPA were rather heavy on amphibole asbestos exposure.

Q Did they have mixed fiber environments?

A Mixed fiber environment, but heavy on the amphiboles.

Q Okay, did they include both chrysitolite and amosite cohorts?

A Some of them had chrysitolite also -- amosite and chrysitolite.

Q Dr. Moolgavkar, what is the relevance of trying to

calculate the doubling-dose information for different fiber types of asbestos?

A Well, I would say it's a lot more important for mesothelioma than for lung cancer. But, as I said earlier, there is some indication that for lung cancer the amphiboles are maybe five to ten times more potent than chrysotile in causing risk -- in increasing the risk of lung cancer. For mesothelioma it's a lot more important because the generally reported figure in Hodgson and Darnton is that hemocyte is about 100 times more potent than chrysotile and that chrysitolite is something like 500 times more potent than chrysotile.

Q Let's move then to mesothelioma and talk about your -- the doubling-dose estimates that you made with respect to mesothelioma. Now, before we actually get to the results of your estimates, you indicated earlier that it's more difficult to estimate the doubling-dose for mesothelioma, and could you explain why that's so?

A Well, as I said, it's more difficult because it's a two-step process. You cannot investigate the relative risk directly so what you need to do is to use an absolute risk model that gives you the number of mesotheliomas for some kind of asbestos exposure.

MS. HARDING: Could you show 2257 please?

A So the absolute risk model that I like to use is Peto's

formula which was used both by EPA in 1986 and by Berman and Crump in 2003 and this is the basic form of this mathematical expression. I Sub-M is the incidence of mesothelioma at Dime D where D is the duration of exposure to asbestos. F is the fiber concentration given to you in fibers per milliliter or cubic centimeter. K Sub-M is a constant that depends upon fiber type. So it's a constant that depends upon whether you're dealing with chrysotile, or chrysitolite or amosite. And D is the duration of exposure in years.

So, what one has to note from this formula, a couple of things. One, in terms of F, the fiber concentration, the incidence is linear. If you increase the fiber concentration from say one fiber per cc to two fibers per cc while keeping the duration constant, you will double the risk of mesothelioma. However, there is a strong non-linearity in duration of exposure. So let's ignore that -- pretend for the moment that thaat creates a little bit of difficulty in understanding. That's the latency or the lag period in that formula. However, let's look at if duration of exposure is 11 years and you increase that to 12 instead of 11, then your risk of mesothelioma will go up, not to twice the amount but will go up eight fold because of that cubic term that duration of exposure is multiplied by.

Q Have you prepared a slide to help explain the appropriate method for modeling doubling-dose for mesothelioma?

A Yes, I have a -- I think I have a slide that shows --
Q Yes, 2258.
MS. HARDING: Could you show 2258 please?
A Yeah, this shows what happens with the Peto formula. And what you can see on the right-hand side of the panel on the left you see that pretty sharp, straight line. That is how the risk increases with fiber concentration.
Q I'm actually going to walk over to make sure we're talking about the right thing. This line here?
A That line --
Q Is that the line you're talking about there?
A Yes.
Q Okay, and what does that line -- why is that line important, what does that tell you about modeling doubling-dose for mesothelioma?
A That line shows how the incidence of mesothelioma increases with the concentration of fibers. Now --
Q And how would you describe that?
A That's linear. It's a straight line. Now, if you look at the other axis and see that curved line going up steeply, that's how the incidence increases with duration of exposure. So it's very clear that duration of exposure is much more powerful in determining risk than in intensity of exposure. I have a simple example up there in that Post-it type note, so if you look at an exposure of four fibers per cc for ten years so

that's a cumulative exposure of 40 fibers per cc years, that yields an incidence of mesothelioma of 40 cases per million per year. Now, if on the other hand, you look at 148 fibers per cc for three years, this is, of course, an extremely high exposure concentration, 148 fibers per cc for three years, that yields also the same risk. It's also 40 cases per million per year. And on the -- what you can see is the first scenario represents a cumulative exposure of 40 fibers per cc years. The next scenario, the second one represents a cumulative exposure which is ten times larger and yet they both yield the same risk.

What this is saying is that it is inappropriate -- to treat the risk of mesothelioma has been linear in cumulative exposure. It is linear in concentration. It is non-linear in duration. And because cumulative exposure consists of both concentration, is made up of both concentration and duration of exposure, cumulative -- the risk of mesothelioma is not linear with cumulative exposure.

Q I want to just make one clarification point. I pointed to this line over here as describing the line -- the linear line that describes the response for concentration of asbestos, is that right?

A Yes.

Q And it's also probably better reflected on this second graph right here, is that right?

A Yes.

Q Okay. Now, with all of those methodological considerations in place, did you then calculate the doubling-dose for mesothelioma and asbestos exposure?
A Yes, I did.
MS. HARDING: Okay. Could you show 2260 please?
A Yes. So, I present three estimates of doubling-dose here assuming a 45-year duration of exposure. So the duration of exposure is chosen to be the one that OSHA usually considers. For mixed fibers, the case of them is reported in Nicholson's EPA document. That's the constant that one requires to use the Peto formula and that is reported to be ten to the minus eight. And using that K Sub-M and assuming a 45-year exposure duration, the cumulative exposure that leads to the doubling of risk is 3.2 fiber per mil years.
Q Dr. Moolgavkar, if you had used a lesser cumulative exposure in your calculation, would that have lowered the doubling-dose that you found?
A I don't understand the question.
Q Okay, all right, maybe I didn't understand. But, the duration of exposure you discussed earlier, you said that it was important. It was more important than concentration.
A Right.
Q Okay, and so I'm just trying to understand -- or actually, maybe -- let's -- I'll move on. The Libby Miners -- what was the doubling-dose for Libby miners?

A Okay. For the Libby miners now I have to use another assumption made by EPA in 1986 because for the Libby miners, K Sub-M is not available. Nobody has really derived a K Sub-M for tremolite fibers or the Libby-type fibers. But one of the assumptions made in EPA 1986 is that there is a fixed ratio between the potency factor for lung cancer and the potency factor for mesothelioma. And so we have the potency factor K Sub-L from the McDonald 2004 study which has found .0036. We use the EPA assumption that K Sub-M is K Sub-L divided by ten to the sixth and come up with the estimate of K Sub-M for Libby fibers which is found .36 times ten to the minus eight. So then using that K Sub-M, the cumulative exposure is 8.9 fiber per mil years.

Q Okay. And, again, you used cumulative exposure in that estimate, as well?

A Well, there's cumulative exposure, but assuming 45 years of exposure. Okay? So the exposure concentration would be 8.9 divided by 45.

Q Okay, now I understand. With respect to chrysotile, did you calculate a doubling-dose for asbestos?

A Yes, the chrysotile doubling-dose comes from Berman and Crump 2003 who present a separate K Sub-M which is what we need for chrysotile and that is reported to be 0.04 times ten to the minus eight. And with that K Sub-M, the cumulative exposure that doubles dose, again, assuming a 45-year exposure duration,